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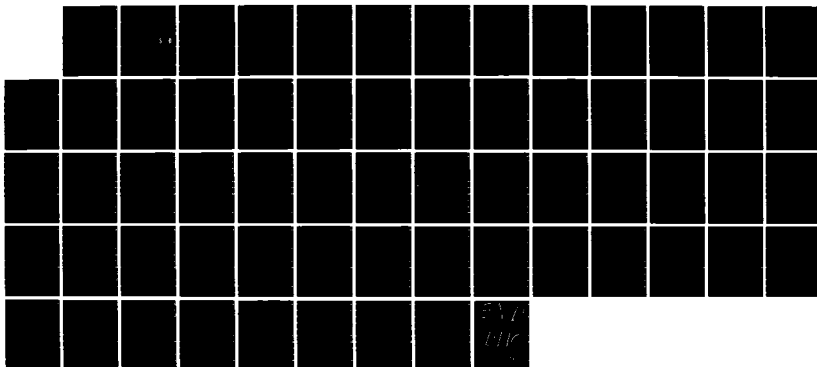
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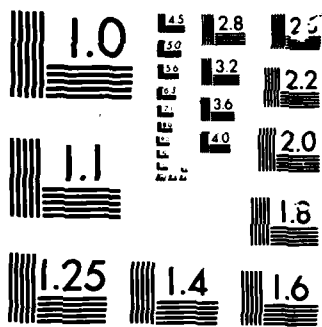
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COORDINATION MECHANISMS IN FAST HUMAN MOVEMENT --  
EXPERIMENTAL AND MODELLING STUDIES

FINAL COMPREHENSIVE REPORT

Walter Kroll  
William L. Kilner

15 July 1984

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number) Result summaries are provided for the modelling and experimental studies of coordination mechanisms in fast human movement. Both isotonic and isometric exercise regimens were used to produce two different levels of fatigue in the agonist or antagonist muscle groups. The triphasic EMG pattern associated with fast ballistic movement was shown to be affected chiefly by a lengthening of the agonist motor time. Faster movement times were produced by antagonist muscle fatigue and slower movement times by agonist muscle fatigue. Vibration was		

shown to be ineffective in altering the movement time and did not affect the learning progress. After development of a mathematical model to accurately predict movement time by relevant EMG measures, a patterned electrical stimulation technique was developed which simulates actual central nervous system innervation of the involved muscle groups. Application of patterned electrical stimulation was shown to produce predicted changes in the triphasic EMG pattern and improve the speed of limb movement by artificial means. Patterned electrical stimulation was not superior to actual physical practice at its present stage of development. Based upon demonstrated improvements in limb movement speed produced by patterned electrical stimulation, a reverse loop sensory imparted learning theory was formulated. Sensory imparted learning is a new form of motor learning for which current motor learning theory has no explanation.

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## Abstract

The Final Comprehensive Report presents abstract summaries of the ten major investigations conducted on Grant DAMD17-80-C-0101. The set of investigations studied the basic neuromuscular coordination mechanisms involved in a rapid elbow flexion movement and developed suitable mathematical models to explain the interaction of these basic coordination mechanisms. The speed of movement was analyzed in a biomechanics mode assessing displacement, velocity, acceleration, point of inflection between acceleration and deceleration, and total speed of movement. The mathematical modelling efforts incorporated these biomechanical parameters and interfaced them with the neurophysiological parameters and central nervous system circuitry, and extended the interface to include viscoelastic properties of the muscle, activation delays, and neuronal pools.

Separate facets of the total approach considered the neuromotor mechanisms of coordination in loaded and unloaded elbow flexion movement; changes in control mechanisms due to practice and learning effects; changes in control mechanisms due to local muscular fatigue induced by isometric and by isotonic exercise in the agonist and in the antagonist muscle groups; feasible training and practice regimens involving artificial means of enhancing beneficial changes in control mechanisms; and development of mathematical models to explain in functional terms ways in which the adaptive mechanisms can account for changes in basic coordination skill and breakdown of coordinated skill due to local muscular fatigue.

The series of studies incorporated research protocols from three usually distinct areas of activity: neurophysiology, biomechanics, computer science and mathematical modelling. Results included the

first valid predictors of limb movement speed, and a functional electrical stimulation technique capable of imparting coordinated limb movement essentially equivalent to actual physical practice.

## INTRODUCTION

Whether the performance task involves the total body as in gymnastics, or only a part of the body as in playing a piano, skilled performance is characterized by a common set of descriptors: smooth, efficient, effortless, coordinated. By now it is recognized that application of force, accurate movement of the body and limbs in a prescribed pattern, and proper speed of execution are the essential ingredients in all skilled human movements. Indeed, skill, coordination, and timing can be considered synonymous terms: the proper amount of force must be applied to produce accurate movement of the body and limbs in a prescribed pattern at appropriate speed to produce efficient execution of a specified task.

Muscular coordination involves the participation of many muscles that essentially act in cooperation with each other to apply force and produce movement, or in opposition to each other to inhibit force application and impede movement. In effect, the properly timed contraction of muscles at specific force levels involving the agonist, antagonist, and synergist muscles must be done in a sequential order for movement to even occur and for efficiency of movement to be possible. The initiation of a movement, control during the movement, and the ending of a movement all depend upon basic neuromotor control mechanisms. It becomes obvious, therefore, that the timing of force application by involved muscle groups is an inherent feature of skilled, coordinated human performance.

One of the more persistent lines of investigation dealing with coordinated movement and application of muscle force has been that dealing with maximum speed of limb movement. Since the force required to cause

movement of a limb is proportional to the distance travelled over time squared,  $F = 2md/t^2$  or  $F = ma$ , it was believed that the greater the static strength of a muscle ( $F$ ), the greater the speed of limb movement which could be produced. When studied in humans, however, the maximum speed of limb movement bore no relationship to the maximum isometric strength of the agonist muscle group.

Although the study of maximum strength of an agonist muscle and maximum speed of limb movement has gone on for several decades, the present status of our understanding of this basic coordinated movement pattern remains painfully inadequate. An enormous amount of evidence exists in support of the untenable notion that human movement does not obey the basic principle of physics that  $F = ma$  since maximum strength of an agonist muscle simply does not correlate with maximum speed of limb movement. Henry and Whitley (1960), Henry (1960, 1962), Clarke (1960), Smith (1961a, 1961b, 1963, 1969), Rasch (1963), and Nelson and Fahrney (1965), for example, all studied the speed of various limb movements and reported non-significant correlations with maximal strength of the agonist muscles involved in producing the movement.

Our laboratories have approached the problem differently and are now able to contend that agonist muscle strength does not correlate with maximum speed of movement because the neuromotor coordination mechanism has been overlooked by past researchers. Any attempt to understand the mechanism involved in fast, ballistic, coordinated movement must consider the sequential timing of activation of agonist AND antagonist muscle groups which control the application of force and produce the resultant movement.

Muscles that are responsible for causing a specific action are known as agonist muscles or prime movers; antagonist muscles produce an action opposite to the action of the prime movers. In any movement of a limb, the force exerted by the prime movers must be large enough to overcome the inertia of the limb and the force applied by the antagonists. With the prime movers in control, movement of the limb occurs as characterized by displacement and acceleration. Limb movement decelerates when the force applied by the antagonists is large enough to overcome the force of the prime movers. Most biomechanical analyses of limb speed of movement have carefully measured acceleration, velocity, point of deceleration, and total movement time. Unfortunately, the biomechanical approach seldom, if ever, sought an explanation of the motion being analyzed in terms of mechanism. Traditional speed-strength investigations have limited themselves to the study of the relationship between maximum strength of agonist muscles and limb speed of movement parameters. For reasons unknown, the maximum strength of the antagonist has been ignored. Even more surprising, the sequential order of muscle activation--which in effect produces and controls movement--has been similarly overlooked.

As long ago as 1677 Descartes postulated that the interaction between muscles was regulated by a series of valves which adjusted the flow of animal spirits into flexor and extensor muscles. The question of the actual interaction between agonist and antagonist muscles in humans has remained a key issue in the neurophysiology of movement. Boulogne (1867) and Winslow (1732) believed that agonist and antagonist muscles contracted simultaneously while Bell (1823) and Pettigrew (1925) contended that the contraction of one muscle was accompanied by relaxation of its antagonist.

Later work by Beaunis (1889) and Demeny (1890) utilized kymography and supported simultaneous contraction of agonist and antagonist. Sherrington (1906), however, promulgated his famous principle of reciprocal innervation and showed that contraction of an agonist was accompanied by inhibition of the antagonist in laboratory preparations. Tilney and Pike (1925) studied many different actions in the upper limbs and were unable to verify the principle of reciprocal innervation in voluntary human movement as they showed both agonist and antagonist active at the same time.

The first electromyographic investigation of the problem by Golla and Hettwer (1924) and later electromyographic studies by Stetson and Bouman (1935), Hudgins (1939), and Bierman and Ralston (1965) suggested that the type of movement being studied was important. Antagonist muscles were usually found to co-contract during slow controlled movements and found not to co-contract during fast ballistic movements. Additional research on the interaction between agonist and antagonist muscles in voluntary movement supports, in general, the phenomenon of co-contraction and that a relationship exists between the speed at which a limb is moved and the sequential order of muscle firing patterns.

A better understanding of the neuromotor control mechanisms involved in limb speed of movement came about when the research methodology of biomechanical analysis of motion was combined with the research methodology of the neurophysiology of movement. The first comprehensive study assessing the neurophysiology and the biomechanics of limb movement was done in our laboratories as part of a Ph.D. dissertation (Lagasse, 1975). By monitoring the time of arrival of nervous impulses to the biceps and triceps

brachii muscles, the initiation of movement, velocity, acceleration, and total speed of movement, Lagasse was able to show that the sequential order of agonist-antagonist muscle activation was a significant predictor of speed of movement; the multiple R of .68 being the highest correlation ever shown between limb speed of movement and a criterion measure related to a probable mechanism. The addition of acceleration time, which alone correlated with speed of movement,  $r = .79$ , resulted in a multiple R of .90. Lagasse also showed that practice influenced the timing and coordination of the neuromuscular control mechanism, largely by a change in the agonist-antagonist sequential innervation pattern.

Individuals capable of the fastest speed of movement do not co-contract agonist and antagonist muscles simultaneously to the degree that the slower subjects do. Marked co-contraction, therefore, may characterize poor neuromotor control while definite agonist-antagonist contraction patterns with a minimum of overlap may characterize skilled neuromotor control. Changes in the agonist-antagonist contraction pattern, furthermore, may actually constitute a pure measure of motor learning reflecting an inherent nervous system capability to learn which might very well be related to a number of other motor performance qualities including motor educability.

#### Statement of the Problem

The investigations conducted studied the muscular coordination mechanisms (as assessed by the sequential activation order of agonist and antagonist muscles) involved in a rapid elbow flexion movement and developed suitable mathematical models to explain the interaction of these basic coordination mechanisms. The speed of movement was analyzed in a biomechanics mode assessing displacement, velocity, acceleration, point of inflection between

acceleration and deceleration, and the total speed of movement. The mathematical modelling incorporated these biomechanical parameters and interfaced them with the neurophysiological parameters and central nervous system circuitry, a part of the basic coordination mechanism, and extended the interface to include viscoelastic properties of the muscle, activation delays and neuronal pools. Separate facets of the basic approach considered:

1. The basic neuromotor control mechanisms of coordination in unloaded and loaded elbow flexion movement.
2. Changes in the neuromotor control mechanisms due to practice and learning effects.
3. Changes in the neuromotor control mechanisms due to local muscular fatigue induced isotonically and isometrically in the agonist and in the antagonist muscle groups.
4. Feasible training and practice regimens involving artificial means of enhancing beneficial changes in the neuromotor control mechanisms based upon known physiological actions of such stimuli as vibration and patterned electrical stimulation as suggested by leads provided by the mathematical modelling.
5. A mathematical model to explain in functional terms ways in which adaptive mechanisms can account for basic coordination skill and breakdown of coordinated skill due to local muscular fatigue.

#### EXPERIMENTAL METHODOLOGY

The basic methodology and appropriate apparatus were developed in preliminary pilot work and in a Ph.D. dissertation by one of our students. The movement studied was forearm flexion from a starting position of 160 degrees to a final position of 90 degrees. Details of the subject posture



and anatomical considerations are based upon the work of Person (1958) and Wilkie (1950) who outlined a set of criteria for selection of a suitable movement to be studied. A class B motion (Bailey and Presgrave, 1958) was chosen in which the motion is volitionally stopped requiring a definite action of the antagonist.

Key features of the apparatus include a potentiometer with appropriate integrated circuitry attached to the axle of a lever arm which is moved by the subject's forearm during elbow flexion speed trials. The circuitry provides output on displacement, velocity, acceleration, and point of inflection between acceleration and deceleration. Separate circuitry provides a measure of total movement time through the prescribed distance of elbow flexion. The sequential order of agonist-antagonist muscle activation was assessed via skin electrodes on the biceps brachii (long head) and the triceps brachii (medial head) and displayed on a storage oscilloscope.

A critical measure of the motion pattern is the specific point of inflection where limb acceleration is replaced by limb deceleration. To produce an angular acceleration ( $\alpha$ ) of the forearm and hand segment, the net torque ( $T$ ) around the axis of rotation ( $o$ ) must equal the product of the second mass moment of inertia ( $I$ ) of the forearm and hand segment and the angular acceleration of the segment. Counter clockwise movement during limb acceleration occurs in the presence of a positive net torque ( $T = I\alpha$ ) producing a resultant muscle force ( $F_m$ ). During limb deceleration of the same movement, the net torque is negative ( $T = -I\alpha$ ) and the force vector ( $F_m$ ) is in the direction of forearm extension. The acceleration-deceleration point of inflection represents the change in resultant muscle action between agonist and antagonist muscle control.

Fatigue. Two levels of exercise intensity were employed in an attempt to produce different rates and intensity of local muscular fatigue. It was reasonable to assume that the development of fatigue using a heavy load and fewer repetitions in isotonic exercise would selectively fatigue the fast twitch muscle fibers more than the slow twitch muscle fibers while use of a lighter load and a greater number of repetitions would more selectively fatigue the slow twitch muscle fibers. The isometric exercise regimens of (a) 5-second contractions and 5-second intertrial rest periods, and (b) 5-second contractions and 20-second intertrial rest periods have been shown to produce significantly different fatigue patterns as far as rate of fatigue, and significantly different amounts of strength loss over the 30 trial exercise series (Kroll, 1974, 1973, 1968).

Informed consent. All subjects used in this investigation were in good health as determined by medical clearance either from the University Health Service or from a personal physician. In accordance with the General Guidelines on the Rights and Welfare of Human Subjects approved by the Faculty Senate of the University of Massachusetts-Amherst on May 11, 1972, an informed consent document (see Appendix B) was presented to each subject for signature. This procedure is in compliance with the policy formulated by the U.S. Department of Health, Education, and Welfare and is described in detail in Section 6.1.0 through 6.5.0 of Senate Document 72-061. The Department of Exercise Science also endorses and complies with the Policy Statement Regarding the Use of Human Subjects and Informed Consent document formulated by the American College of Sports Medicine published in the Fall, 1975 issue of Medicine and Science in Sports, and the National Research Act of 1974 (Public Law 93-348).

## TESTING SCHEDULE

### Phase I. Unloaded Movement Coordination

Subjects performed 40 trials daily of rapid elbow flexion speed of movement. Four to six days of practice were necessary for learning to occur and provided basic data on the changes in neuromotor control mechanisms and motion characteristics. Following establishment of stabilized, well practiced performance, local muscular fatigue was induced as per the following exercise regimens:

#### 1. Agonist Muscle Fatigue: Isometric

- a. Five second maximal isometric contractions of elbow flexion with five second intertrial rest periods over 30 trials.

In series two, the intertrial rest periods were 20-seconds.

#### b. Agonist Muscle Fatigue: Isotonic

Six bouts of isotonic elbow flexion. During each bout subjects performed repetitions at a load equal to that necessary to produce a six repetition maximum (6-RM) or a 20 repetition maximum (20-RM) bout. Preliminary testing on each subject during the baseline stabilization days established the individual loads to be used to produce a high intensity (6-RM) and low intensity (20-RM) fatiguing exercise regimen.

#### 2a. Antagonist Muscle Fatigue: Isometric

- a. Same protocol as 1a above.

#### b. Antagonist Muscle Fatigue: Isotonic

- b. Same protocol as 1b above.

One of the purposes of inducing high and low intensity local muscular fatigue via isometric and isotonic exercise regimens on the agonist and

on the antagonist muscle groups separately was to ascertain the role of changes in peripheral muscle feedback to higher nervous system centers and determine if programmed central commands for a fast ballistic movement can be altered. Information from this aspect of the study was deemed to be of considerable value in planning the exact modalities employed in Phase III of the proposed investigation.

#### Phase II. Loaded Movement Coordination

The same testing schedule proposed in Phase I was replicated during Phase II for elbow flexion speed of movement against a load. In order to insure identical loading for subjects with different limb lengths, an inertial loading technique was utilized. As discussed by Stothart (1970), the natural moment of inertia differs by placement of light weights at long distances and heavy weights at short distances from the fulcrum point. The present study utilized a fixed weight for all subjects and varied the distance to comply with the condition of imposed amount of inertia. One load condition was two times the natural moment of inertia and load condition two was five times the natural moment of inertia.

#### Phase III. Experimental Manipulation of the Neuromotor Control Mechanisms

Phase III depended heavily upon the results of the first two phases of the proposed study. It was shown that the basic neuromotor control mechanism can be changed through practice/learning and/or altered by local muscular fatigue, then a search for means by which to induce changes in the basic coordination mechanism could be sought. The mathematical modelling results from the basic data of Phases I and II produced fruitful leads for such an endeavor.

As a direct result of research completed during Years 1 and 2, the reverse loop theory of motor learning was formulated and presented at various conventions and invited talks. The purpose of such informal presentations was to elicit reactions and critiques of the theory for resolution in further studies. We have shown that improvement in limb movement speed can be produced by patterned electrical stimulation of relevant muscle synergy action patterns without any actual physical practice. Such limb movement speed improvements are, furthermore, quantitatively similar to actual physical practice. Changes produced in the electromyographic patterns for limb movement tasks follow the patterned electrical stimulation protocols. Improvements in neuromotor coordination mechanisms and limb movement speed produced by the patterned electrical stimulation are presumed to be due to sensory imparted learning, and constitute experimental evidence for the reverse loop theory of motor learning. Both the reverse loop and sensory imparted learning theories promise to alter radically present motor learning theories and result in an enhanced understanding of motor skill acquisition in general.

In order to validate properly the sensory imparted learning and reverse loop theories, it was planned to apply patterned electrical stimulation protocols based upon different combinations of the identified neuromotor coordination mechanisms described in our mathematical model of fast limb movements (Kilmer, Kroll, & Congdon, 1982). During Year 3 we proposed, therefore, a series of studies designed to elicit relevant experimental tests of the model. Briefly, the series of studies involved patterned electrical stimulation to produce changes in limb movement speed and neuromotor coordination mechanisms based upon different protocols:

- Series 1. Manipulation of the three basic neuromotor coordination mechanisms.
- Series 2. Study of high frequency electrostimulation purported by Russian investigators to produce better strength gains than traditional exercise systems. The goal was to determine whether neuromotor coordination mechanisms can be altered by electrically produced strength changes in either the elbow flexor or the elbow extensor muscle groups. If selective strength changes could be produced in the flexor and/or the extensor muscle groups, the proposed mathematical model for neuromotor coordination mechanisms in fast limb movements could be further tested.

Results from the early series of studies suggested that limb movement coordination mechanisms were markedly affected by inertial loading of the limb. In order to investigate this lead, we administered patterned electrical stimulation based upon neuromotor coordination mechanism parameters elicited under different inertial loads. The addition of the inertial load parameter necessitated additional studies over and above those originally planned for Year 3. The high frequency electrostimulators were delayed in shipment due to difficulties in securing the Russian type stimulators from the only U.S. supplier.

#### Overview of Modelling Studies

The purpose of the modelling studies was to gain a fundamental understanding of how well-coordinated, fast, ballistic human limb movements are learned and organized. Without a model, one doesn't have a really precise account of the mechanisms involved in a phenomenon, and so doesn't

know what to look for next. The studies required an adaptive model because the skills to be studied are themselves adaptive. Because of the decade of brain modelling experience of P.I. Kilmer and several others in the COINS Department at the University of Massachusetts-Amherst, and P.I. Kilmer's longstanding technical interest in P.I. Kroll's experimental research on fast movements, the combined experimental and modelling study proved fruitful.

The modelling work was based upon some of the excellent results and techniques developed to date in models of fast elbow flexion (Dijkstra and van der Gon, 1973; Lagasse, 1975), leg shock while running on Harvard University's new indoor track (McMahon and Greene, 1978), stretch reflex function (Houk, 1976), and human eye movement (Bahill and Stark, 1979). Mathematics and computer simulation was employed to study stability, time-delay effects, nonlinearities, the behavior of systems with distributed pools of components, and the use of trainable neurons for optimally controlling nerve and muscle systems. The long range goal was to understand how complex well-coordinated human movements are learned and organized, how they are affected by stresses, and what optimal training methods might be like.

#### Discussion of Modelling Studies

The basic structure of the fast limb movement system investigated by Kroll has already been modelled. Figure 1 outlines this structure for one muscle pair and Figure 2 delineates the model.

One mathematical result we have proved for the model is the following: let  $\Delta = T_\alpha + T$ , the total delay around the  $\alpha - \gamma$  loop, and define  $kA$  as the loop gain,  $G$ . Then for certain values of the model parameters,

if  $u = 0$ , the response  $\phi$  is stable (doesn't oscillate with ever greater magnitude) if and only if  $D/G \Delta$  exceeds a certain constant  $C$ , where  $D$  is the damping factor in the muscle model. Computer simulation of the model has given dramatic confirmation of this result.

The Figure 2 model does not account for the Renshaw-cell controlled silent period of about 50 ms which neurophysiologists have measured in non-humans after maximal  $\alpha$  motoneuronal bursts are commanded from higher motor centers. To compensate for this, we assume that  $J$  in Figure 2 admits an approximately equivalent rate of tension development in the muscle to what is found in the actual case. Later models will address the Renshaw and other effects as outlined in the Yearly "Goals" Section below.

Figure 3 shows an abstract idealization of the optimal ("bang-bang") control of the fast movement sketched in Figure 2a). Such control problems have been solved mathematically (cf., Burghes and Downs, 1975) for the switching time  $t$ , at which full flexor force gives way to full extensor force to brake the movement so as to stop it exactly at  $t_2$  on target  $\phi_T$ . In this case  $t_2 = T$ , the total movement time, because the  $\gamma$  feedback linkage is assumed not to be used at all. In reality, not only is the  $\gamma$  feedback loop always engaged, but also flexor and extensor co-contractions occur, presumably to stabilize the joint and perhaps to prevent excessive forces from damaging the joint. (In an experiment on himself, Kroll quickly conditioned out the initial co-contraction in a fast elbow flexion by electric shock reinforcement. This decreased his movement time by about 20%, but the next day he awoke with a very sore and swollen elbow). Later versions of the Figure 2 model will attempt to explain the possible benefits and modifications of various kinds and degrees of co-contraction.



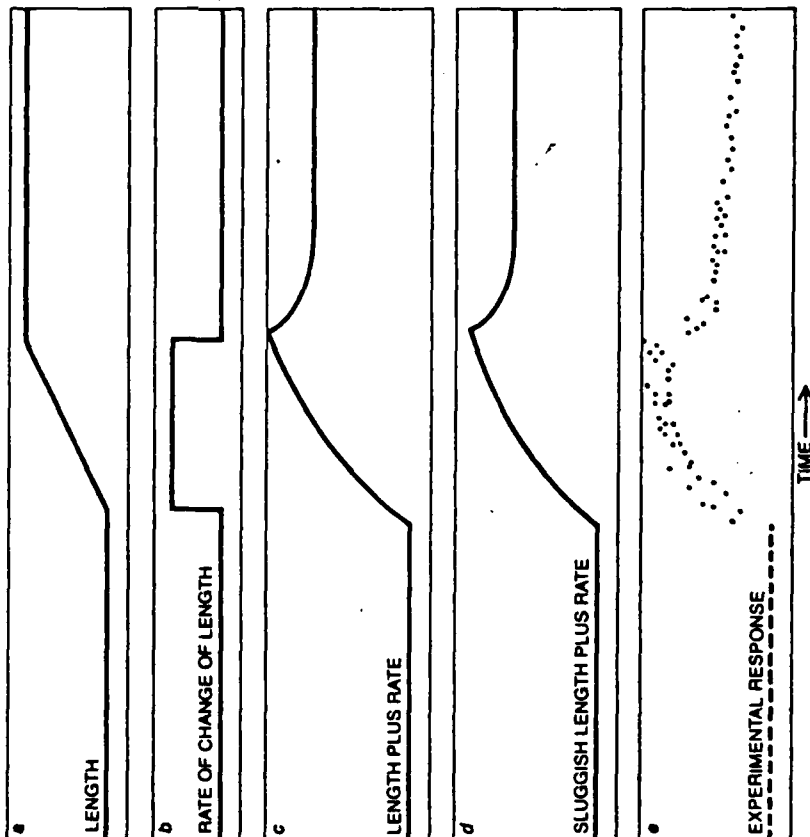
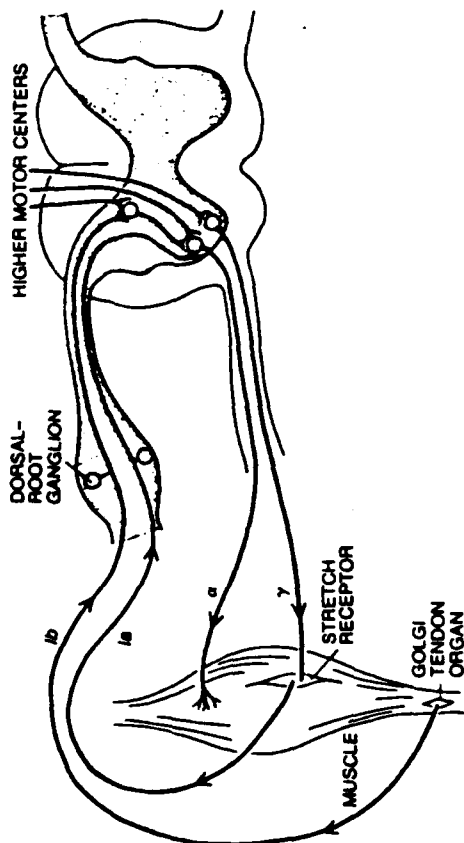


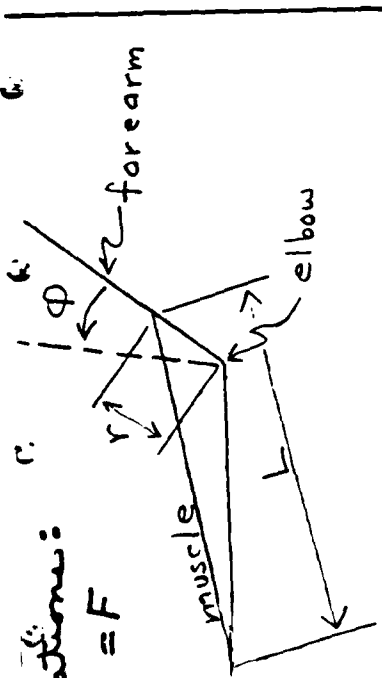
Figure 1. The part of the fast movement system modelled to date for one extensor/flexor pair consists of  $\alpha$  command fibers from higher motor centers,  $\alpha$ - $\gamma$  loops, and associated muscles and tendon organs. We assume that at the outset of a movement, the stretch receptors are quickly reset to the new desired reference length, and that the  $\alpha$  motoneurons maximally excite the antagonist muscles. For the fastest possible movement, the antagonist muscles should not be excited until time to brake the movement in order to stop it at the target position. We assume that the tensor organs help to modify the stretch response in such a way that muscle stiffness (change in force divided by change in length) is essentially constant over most of the movement. We assume further that the rate sensitivity of the stretch receptors shown in a to e underlie much of the force-velocity damping relation we have modeled with a dashpot in the muscle function block of Figure 2. The rest of the damping derives from tendon organ, spinal neuronal, and intramuscular effects.

a) Some metric relations:

muscle tension =  $F$

$$F = I \frac{d^2 \phi}{dt^2}$$

$$\frac{dL}{dt} \approx r \frac{d\phi}{dt}$$



b) The system block diagram

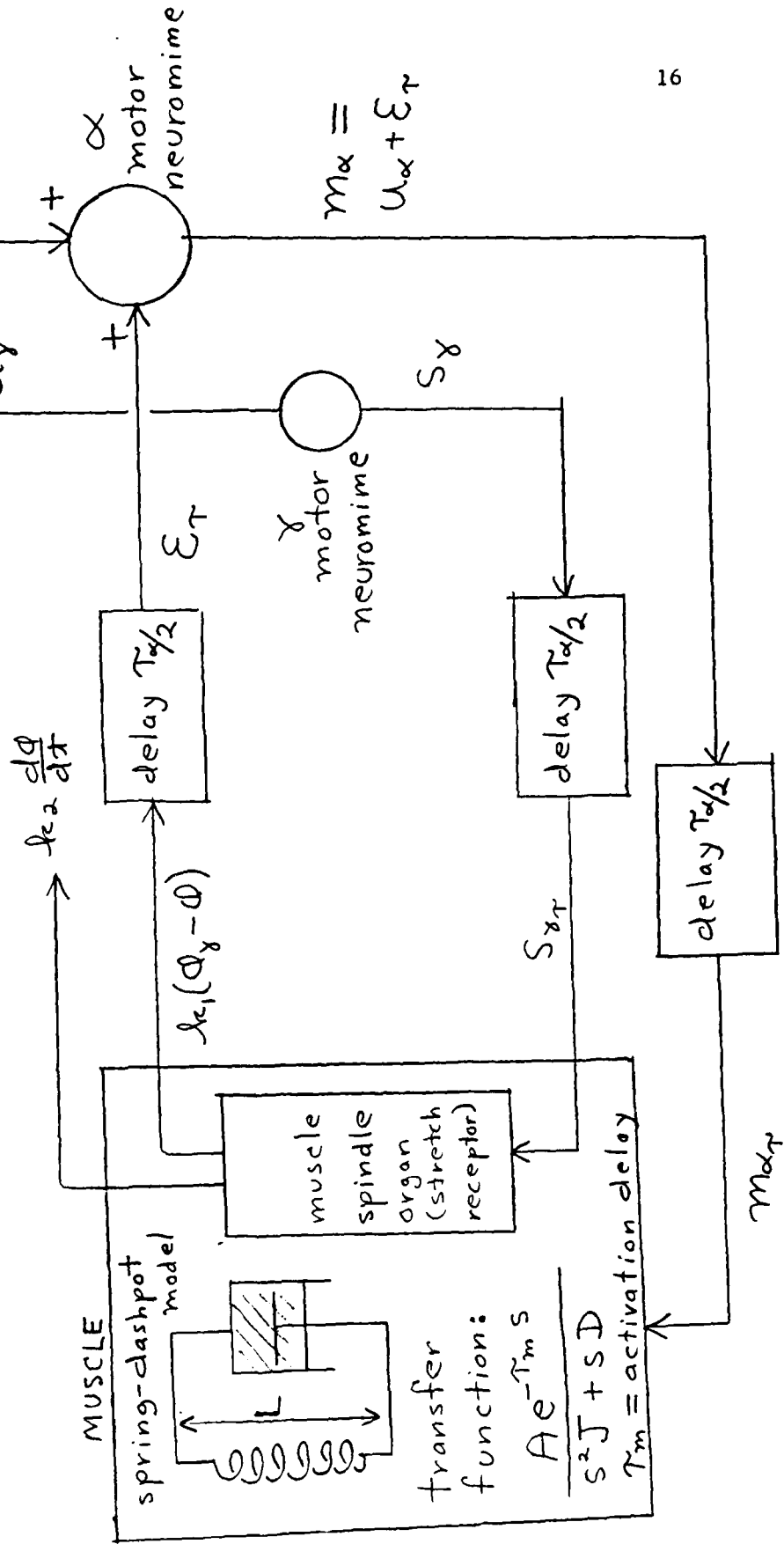


Figure 2. b) shows the analysis block diagram for the first model of fast arm movement schematized for one flexor/extensor pair in a). The initial  $\phi$  is assumed zero; the final  $\phi$  is denoted  $\phi_T$ ; the present reference  $\phi$  in the spindle is denoted  $\phi_Y$ ; flexor magnitudes and directions are positive and extensor ones are negative. The input to the extrafusal muscle on the left of b) is assumed to act as if it served to reset a spring stretched  $x\phi_F$  units from neutral. The resultant muscle force is assumed to equal the force across the dashpot  $D[d\phi/dt]$  plus the tension  $F$  that gives rise to the  $d^2\phi/dt^2$  of the effective inertia  $J$ . The muscle activation delay is denoted  $\tau_m$ , and the  $m_{\alpha_T}$ -to-force conversion factor is  $A$ . The muscle spindle is assumed to act as a linear strain gauge. The equation for  $\phi$  in the Laplace domain is:

$$\phi(s) = \left[ \frac{A e^{-\tau_m s}}{s^2 J + s D} \right] e^{-\left(\frac{\tau_m}{2}\right)s} \left[ u_\alpha(s) + e^{-\left(\frac{\tau_m}{2}\right)s} k_1 (\phi_Y(s) - \phi(s)) \right]$$

The introduction of nonlinearities into this model would necessitate separate subsystems for flexor and extensor.

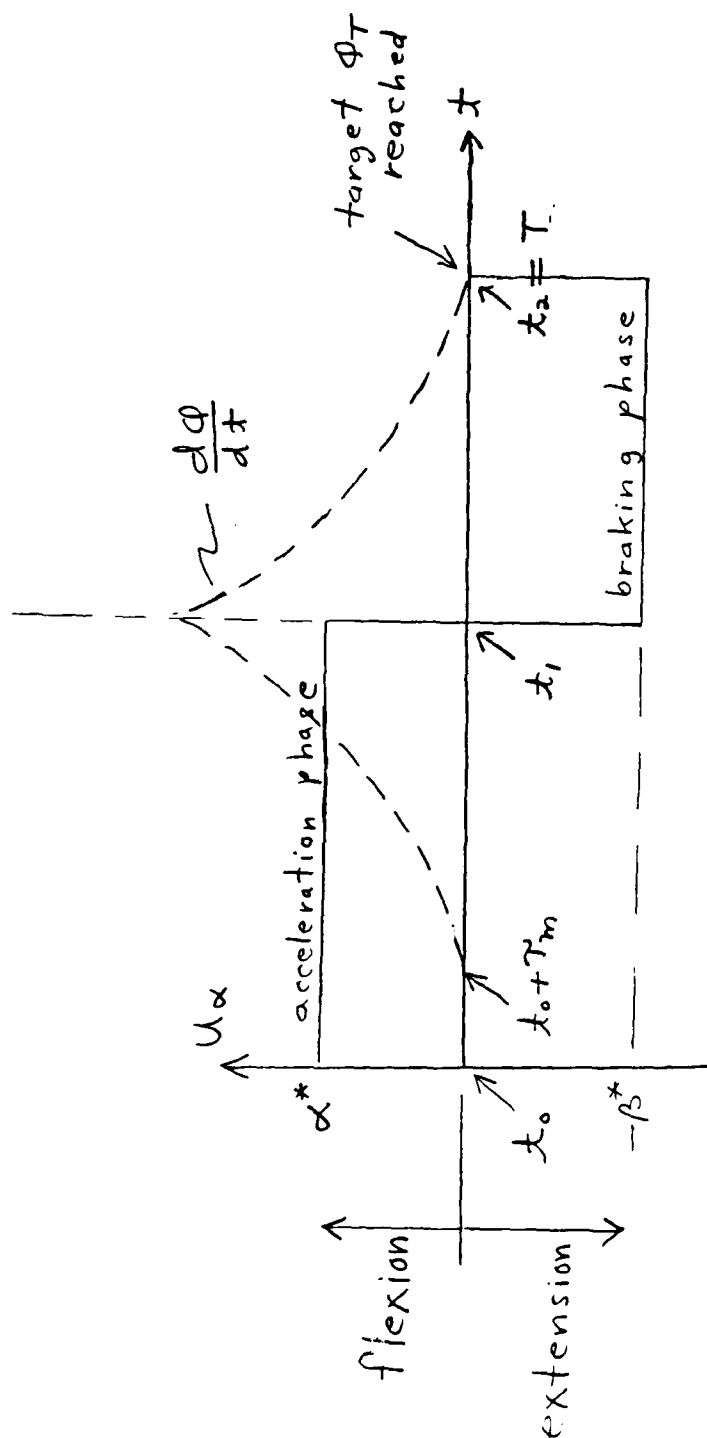


Figure 3. Abstract idealization of the  $u_\alpha$  feed-forward-only (bang-bang) concept of optimal control of a minimum-time movement to the target position  $\phi_T$ .

Figure 4. An outline of the nature of the control problem in Figure 2 when:  $u_\alpha$  is of the form shown in Figure 3; and whenever  $|u_\alpha| \neq 0$ , it is at least several times greater than  $|k_1(\phi_Y - \phi)|$ . Denoting  $d\phi/dt$  by  $\dot{\phi}$ , in Figure 3 during the interval from 0 to  $t$ ,  $\dot{\phi}$  is roughly proportional to  $t$  and  $\phi$  is roughly proportional to  $t^2$ . Thus

$$\frac{\max \dot{\phi}}{\sqrt{\phi_Y}} \approx \frac{C_1 T}{C_2 T} = C_0$$

$$\underbrace{[\ln(\max \dot{\phi})]}_X - \underbrace{[\frac{1}{2} \ln \phi_Y]}_Y - \underbrace{[\ln C_0]}_Z \approx 0$$

assume  $Y \geq 0$

$$\text{That is, } X - Y - Z \approx 0$$

A device that fires an output signal at  $t_1$  in Figure 3 is the heterostat neuromime ~~neuro~~  $N_1$  defined below:

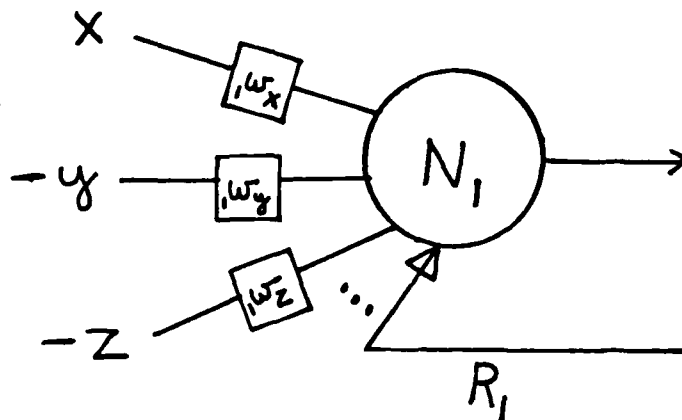
Operation:

If  $w_x X + w_y Y + w_z Z$  exceeds 0,  $N_1$  fires

Training:

$R_1$  trains the  $w$ .

See text for details



Similarly, a heterostat that fires at  $t_2$  in Figure 3 is defined by:

$$u = -\dot{\epsilon}$$

$$v = -\dot{\phi}$$

$$r = k_3 \ln \phi_Y, \text{ for } \phi_Y \text{ of Fig. 3}$$

Operation:

If  $2w_u u + 2w_v v + 2w_r r$  exceeds 0,  $N_2$  fires

Training:

$R_2$  trains the  $2w$ . See text for details

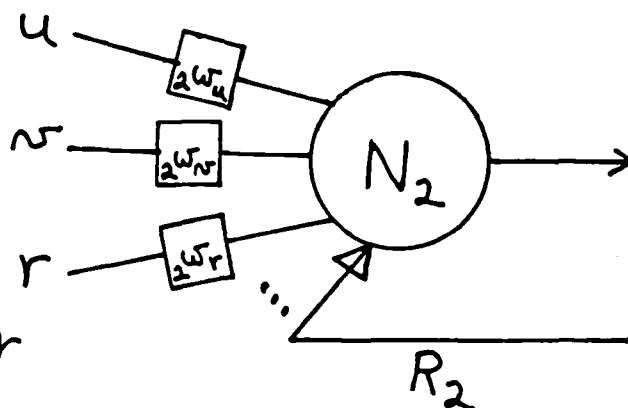


Figure 4 sketches the nature of the Figure 2 control problem when both  $u_\alpha$  and  $u_\gamma$  control is used. The trainable neuromine components in Figure 4 represent our first attempt to model the way that learning to execute a faster movement can be mechanized. Letting  $t$  be the time in Figure 3 that the maximum flexor command  $u_\alpha$  is cut off, and  $t_2$  be the time that the maximum extensor command  $u_\alpha$  is cut off, we show  $N_1$  computing  $t$ , as trained by the  $jw$ -reinforcement signal  $R_1$ , and  $N_2$  computing  $t_2$  as trained by  $R_2$ . Of course when flexor and extensor signal maxima overlap, other  $N_i$  are needed to compute the associated additional  $t_i$ .

A reasonable rule for  $R_1$  is as follows: decrease  $jw_x$ ,  $jw_y$ , and  $jw_z$  each trial by an amount roughly proportional to the duration over which the proposition ( $u_\alpha = 0$  and  $N_2$  has not yet fired) is true. A similar rule for  $R_2$  is: increase  $2w_u$ ,  $2w_v$ , and  $2w_r$  each trial by an amount roughly proportional to the duration over which the proposition ( $u_\alpha = 0$  and  $N_2$  has fired and  $(k_4 |\epsilon| + K_5 |\dot{\phi}|) < k_6$ ) is true, for appropriate values of  $k_4$ ,  $k_5$  and  $k_6$ .

$N_1$  and  $N_2$  are not the old threshold learning devices, but a sophisticated new component now being studied under contract with the Air Force at U. Mass (Barto and Sutton, 1979). This component is called a heterostat, and its inventor, Dr. Harry Klopff, intended it as a better model of real trainable neurons than was formerly available. Its key feature is its retention of articulate traces of recent input stimuli which can later be used to modify its  $jw_i$  memory elements.

Our eventual aim was to understand functionally what happens when a human learns to make his movements faster, better coordinated, and more precise. Granit (1977) says "what is volitional in voluntary movement

is its purpose". Assuming that voluntary movements are built on a reflex basis, our cybernetic modelling studies were able to elucidate some "simpler" voluntary movements.

#### Summary of Results

Detailed reports of the major investigations conducted have been presented in annual reports, published articles, master's theses, and doctoral dissertations. Short summaries of the most important results for each of the major studies conducted will be presented here.

### An EMG-Level Muscle Model for a Fast Arm Movement to Target\*

A model of human muscle action is presented for a maximally fast, large-amplitude forearm movement to target. The inputs to the model approximate the biceps and triceps brachii EMG envelopes over a single movement. The model's output gives the corresponding displacement angle of the forearm about a fixed elbow position as a function of time. The idea of the model is to conceive of both input drives as successions of millisecond input pulses, with each pulse resulting in a muscle tension twitch. Every twitch is amplitude-scaled, parametrically-shaped, and duration-limited as a function of the muscle's contractile history thus far in the movement. The muscle tension at any time  $t$  is the sum of the residual tension levels of all twitches begun before  $t$ . The model was developed and tested with special reference to two subjects: one, according to the model dynamics, was a comparatively slow-twitch type, and the other modelled as a fast-twitch type. Good agreement was found between model output and subject response data whenever the subject's EMG's were "synchronous". The model can be used to characterize each subject's responses by a suite of twitch characteristics. This will enable us to check the accepted but now suspect correlation between muscle biopsy- and performance-determined muscle twitch type.



This is the first attempt we know of to analyze  $EMG(t)$ -to- $T(t)$  transformations where  $T$  is the tension noted in muscles controlling fast, large-amplitude limb movement to a target. The most closely related modelling works to date are: Stein and Oguztoreli (1981)'s small signal linear analysis of the mammalian-neuromuscular and reflex system; Hatze's (1978, 1980, 1981) papers at a more detailed level involving state equations for numbers of motor units in different contractile states; Sakitt's (1980) steady-state spring model for postural changes and Dijkstra et al.'s (1973a, b) original cross-bridge model and analog computer simulation of small amplitude human forearm movements.

Our model attempts to better determine the correlations between: 1) the speed (power) and shape of maximally fast, large-amplitude human arm movements to a target; (2) the proportions of fast- vs. slow-twitch fibers, as determined by muscle biopsy in subjects making such movements; and (3) the physiologically effective twitch speeds involved at different stages of each such movement when performed by highly skilled (i.e., well-practiced) subjects.

Our J-Model should prove useful in at least two ways. First, it should enable us to check further on the presently entrenched but increasingly suspect ideas on the role of biochemically classified fast-twitch and slow-twitch muscle fibers in fast movements such as ours. Some evidence to date suggests that the slow- and fast-twitch fibers of biopsy are not always the slow- and fast-twitch fibers of action. Now, by comparing J-Model simulation with muscle biopsy results, we can produce dynamically valid correlations on this question.

A second use of J-Model should be to aid the design of optimal electrical stimulation patterns for use in limb rehabilitation following strokes and other accidents. This would build on Boucher and Lagasse's (1980) success in transferring a skilled muscle activation pattern for our movement type from "donors" to unpracticed "recipients".

Full details of the above study can be found in the February 1982 Annual Report and in the publication "An EMG-Level Muscle Model for a Fast Arm Movement to Target" published in Biological Cybernetics, 44, 17-26, 1982, by W. Kilmer, W. Kroll, and V. Congdon.

## On the Stability of Delay Equation Models of Simple Human Stretch Reflexes.

Human stretch reflexes (SRs) are often too weak and ineffectual to provide adequate postural regulation or rhythmic movement boosting (e.g. in ankle pushoff at the end of stance phase in fast running). Recent improvements in the methods of artificially enhancing skeletomotor responses, especially in therapeutic regimens, should not be widely employed until the clonus-resisting stability properties of SRs are better understood.

We formulate an idealized linear servo model of a segmentally mediated SR system which includes the often ignored electromechanical coupling delay. For typical closed-loop (delay/gain) ratios, the model is shown to be unstable for all values of loop gain when operating as a position servo, but maximally stable when operating as a velocity servo. We claim that the velocity servo or one of its nonlinear relatives is a better model for some well studied SRs than, for example, Houk's stiff muscle hypothesis. We also present evidence that even feeble and quickly saturating monosynaptic postural servos are always unstable if operated as pure position regulators.

The linearized SSR model is unstable as a position servo, and stable at sufficiently small gains as a velocity servo. We propose that the velocity servo formulation fits Dietz's triceps SR data for falling better than Houk's stiff muscle hypothesis. Some other SRs, such as occur in biceps brachii when an arm in handshake position is suddenly unloaded, should also eventually model best as velocity servos because they are among the more powerful clonus-free SR systems.

Strong analytical and simulation evidence is presented that the pure position servo with one or two long loops added is unstable for all positive gains and delay sets, and that the corresponding velocity servos are stable as long as the sum of the gain-delay products for all loops is less than a fairly tight bound. This suggests that the possibility that all control of postural steadiness is mediated by the following: steady, centrally produced signals; velocity or mixed velocity and position servos (accounting for the 10 Hz. tremor); and volitional saccades.

Full details of the above study can be found in the February 1982 Annual Report and in the publication "On the stability of delay equation models of simple human stretch reflexes" published in the Journal of Mathematical Biology, 17, 331-349, 1983 by W. Kilmer, W. Kroll, and R. Pelosi.

## Riccati Equation Solution for Controllers with Continuous Delays

We reduce the solution of a Riccati equation for infinite-time linear quadratic controllers with continuous delays and  $n$  state variables to the problem of finding scalar parameter values for an integral kernel whose form is completely specified. To simplify the exposition, the reduction is described only for a special case involving 2-dimensional variables, but the method is entirely general. An abstract formulation of Vinter and Kwong is used throughout.

Full details of the above study--which is essentially a mathematical proof-- can be found in the February 1982 Annual Report and in the publication "Riccati equation solution for controllers with continuous delays" published in Systems and Control Letters, 3, 203-209, 1983 by W. Kilmer and W. Kroll

Functional Electrical Stimulation, Sensory Impaired Learning, and Neuromotor Coordination Mechanisms: Relevance to Therapeutic and Limb Rehabilitation Programs.

It has been proposed that the implicit goal of all neurophysiologically based therapeutic exercise systems is to transmit sensory stimulus patterns to the central nervous system which are more intense than that produced without benefit of proprioceptive facilitation. Implicit in such a treatment rationale is the belief that transmission of heightened sensory stimulus patterns to the central nervous system will enhance recovery of lost neurological functions. Various mechanisms for neurological function recovery have been proposed: collateral sprouting, unmasking, reactive synaptogenesis, subliminal fringe neuron activation, reclaiming of ipsilateral nerve pathway control tracts.

Motor command centers in the brain which have been damaged by some pathology can produce only inefficient and incorrect innervation commands to the muscles. Limbs afflicted by some pathological condition can produce only inefficient and incorrect volitional movement patterns. Such inefficient and incorrect volitional movement produces a sensory stimulus pattern which is itself inefficient and incorrect, even if enhanced by proprioceptive facilitation techniques. If recovery of central nervous system functioning is dependent to any degree upon the quality of the sensory stimulus pattern received, the recovery

is likely to be slow and dependent upon gradual improvement in the quality of the limb movement synergy which produces slight sensory stimulus patterns.

If, however, the sensory stimulus pattern transmitted to the central nervous system from an afflicted limb was not an impoverished pattern, but a sensory stimulus pattern representing optimally efficient limb movement, the likelihood of improved neurological recovery might be enhanced. Such a possibility now seems feasible because of developments in the areas of functional electrical stimulation, neuromotor coordination mechanisms controlling fast limb movements, and sensory imparted learning. These relatively distinct areas of research have an obvious relevance to each other, and a blending of their complementary components holds promise for a vastly improved rehabilitation regimen for neurological recovery. Based upon a mathematical model of neuromotor coordination mechanisms for optimally efficient limb movement, functional electrical stimulation can be applied to an afflicted limb which can produce an optimal sensory stimulus pattern for transmission to the central nervous system. If neurological recovery is dependent to any degree upon the quality of the sensory stimulus pattern, receipt of such optimized sensory stimulus patterns should be more effective than any present therapeutic exercise regimen. Since the functional electrical stimulation is applied to a mathematical model for neuromotor coordination mechanisms, a suitable set of criterion measures are

available for assessing true neurological changes from changes in the peripheral musculature.

Full details of the above paper can be found in the February 1982 Annual Report. It was presented at the Neurophysiology Symposium on Movement Disorders, Columbia University, March 16, 1982. A revised version is being prepared for submission to Stroke.



Prediction of Male and Female Isometric Arm Strength  
by Anthropometric Measures

Twenty male and twenty female subjects (ages 17 to 28 years) were measured for maximal isometric elbow flexion and extension strength, upper limb volume, lengths and girths. Female flexion strength was 54.3% and extension strength 58.2% of males. Covariance adjustment for body weight and upper limb volume increased female flexion strength to 78% and extension strength to 73% of males, but males were still significantly stronger. Arm girths and limb volume exhibited larger sex differences than strength measures. Limb lengths were not as important as girths; and body weight and limb volume importance in strength prediction differed in males and females. Multiple R's of .84 to .94 for strength resulted from a full set of anthropometric predictors. Simple measures of limb girths and lengths, with and without body weight, were as good or better than segmental limb volumes in strength prediction. It was clear that: (1) the importance of anthropometric measures as strength predictors differs not only between the sexes but between strength measures as well; (2) male and female differences extend beyond absolute differences in anthropometric measures and includes differences in the relationship of upper limb anthropometric measures; (3) body size factors do not explain adequately the strength superiority of males.

Full details of the above study can be found in the February 1982 Annual Report. The paper has been reviewed and is now being revised for publication in Human Biology.

Gender Differences and the Effects of Isometric Fatigue and Relative  
Isometric Fatigue on the Maximum Speed of Human Forearm Flexion Under  
Resisted and Unresisted Conditions

This investigation addressed the critical need to ascertain gender differences, if any, in the recognized neuromotor mechanisms of intentionally arrested forearm flexion: the sequential order of muscle activation, the bioelectric activity from the biceps and triceps brachii, and the occurrence of the acceleration-deceleration point of inflection.

The maximum speed of forearm flexion, bioelectric coordination mechanisms, and measures of maximum voluntary contractile force in the forearm flexors and extensors were assessed in twenty-four college aged men and women. All measures were collected under the normal inertial resistance condition and under two increased inertial resistance conditions.

The influence of isometric fatigue imposed separately and concurrently on the forearm flexors and extensors was assessed relative to the neuromotor coordination mechanisms under investigation. Comparative data were collected before and after each fatigue regimen.

Practice effects

Contrasting practice day 1 with practice day 4, the men decreased movement time to maximal displacement by 13.5 msec., 14.3 msec., and 8.1 msec. under load conditions 0, 1, and 2, respectively. The women decreased movement time to maximal displacement by 33.1 msec.,

16.3 msec., and 15.9 msec. under load conditions 0, 1, and 2, respectively. In a similar comparison, during the first ninety degrees of forearm flexion, the men decreased movement time by 4.0 msec., 9.4 msec., and 0.4 msec. under load conditions 0, 1, and 2, respectively. The women decreased movement time by 19.2 msec., 14.6 msec., and 12.3 msec. under load conditions 0, 1, and 2, respectively. Clearly the women experienced more pronounced practice effects, enabling them to narrow the difference between the genders, particularly during the first ninety degrees of flexion. The women were not as successful in narrowing the difference between the genders in movement time to maximal displacement due to accuracy deficiencies. They were less accurate and, therefore, flexed further past the ninety degree target resulting in increased movement times.

Gender influences were also revealed in acceleration time to maximal displacement and in acceleration time during the first ninety degrees of forearm flexion. In both cases the women maintained the longer acceleration times, under all load conditions. The women exhibited a significantly longer time to the second burst from the triceps brachii. This fact could partially account for the smaller difference between genders for movement time during the first ninety degrees of forearm flexion. The delay, in the second triceps burst, allowed for the longer acceleration time and, consequently, the decreased movement time. The delay also had implications for the reduction in accuracy, the women did not seem able to couple the delay, in the second triceps burst, with a contraction sufficient

enough to brake the forearm near ninety degrees of flexion. The duration of the first biceps burst was also subject to gender influences, as the women required a longer biceps duration to overcome the initial inertial of the forearm.

#### Influence of induced fatigue

The variance analysis of the criterion measures after the imposition of the fatigue regimens yielded significant gender, load condition, and regimen effects for movement time, during the first ninety degrees of forearm flexion; time to second triceps burst, also during the first ninety degrees of forearm flexion; second triceps duration, to maximal displacement; and the slope of the second triceps burst EMG. Agonist fatigue regimens, 5:5, 5:10, and both 5/5:0 regimens produced a delayed second burst from the triceps brachii which translated into increased movement times. Antagonist fatigue regimens, 5:5 and 5:10, produced earlier and longer second bursts from the triceps brachii with a resultant trend toward decreased movement times.

The imposed fatigue regimens also produced significant changes in movement time to maximal displacement; acceleration time to maximal displacement; first biceps burst to second triceps burst latency; second triceps burst to maximal acceleration; and second triceps burst to zero acceleration latency. Agonist fatigue regimens combined increased acceleration time, increased latencies for the first biceps burst to second triceps burst, decreased latencies for second triceps to zero acceleration latency, and increased latencies for second triceps to maximal acceleration with resultant increased movement time.

Antagonist, 5:5 and 5:10, fatigue regimens combined decreased acceleration time, decreased latencies for the first biceps burst to second triceps burst, increased latencies for second triceps to zero acceleration, and decreased latencies for second triceps to maximal acceleration with resultant trends toward decreased movement times. All of the criterion measures, which were significantly altered by the imposed fatigue regimens, were directly associated with the second burst from the triceps brachii. Once again establishing the key role of the second triceps burst in the speed of human forearm flexion.

#### Movement time prediction formulae

A factor analysis was conducted on the criterion measures, to maximal displacement, including isometric flexion and extension M.V.C. measures. The most heavily loaded variable in each factor and a grouping variable, to account for gender, were submitted to stepwise multiple regression analysis. The resulting predictive equations, for each load condition, showed that the predictive value of acceleration time alone ranged from  $r = .88$  to  $r = .92$  or 77 to 85 percent of the variance associated with movement time to maximal displacement. Although the gender variable was selected third in the prediction formulae for load condition 1 and 2, its inclusion in the prediction formulae did not attain significance at the .05 level of confidence. The predictive value of the ratio between the total biceps EMG and the total triceps EMG was revealed, under all load conditions. The inclusion of this criterion measure, as the second or third variable, accounted for a

significant portion of the variance associated with movement time and, therefore, enhanced the predictive power of the formula.

In an attempt to quantify the predictive power of the neuromotor mechanisms studied in the present investigation, another stepwise multiple regression analysis was conducted. Acceleration time was removed from this analysis since it was not considered a neuromotor mechanism. The set of predictors available for selection included: first biceps motor time, second triceps motor time, second triceps duration, first biceps burst to second triceps burst latency, slope for the first biceps burst EMG, slope for the second triceps burst EMG, ratio between first biceps burst EMG and second triceps burst EMG, ratio between total biceps EMG total triceps EMG, flexion M.V.C., and extension M.V.C.

The analysis for Load 0 with acceleration time and other selected criterion measures removed from the set of available predictors. The first biceps burst to second triceps burst latency was selected first, whenever it was available, it accounted for 61 percent of the variance associated with movement time to maximal displacement. When the first biceps burst to second triceps burst latency was removed two measures related to the second triceps burst were selected. However, the slope for the second triceps burst EMG and the second triceps motor time combined just exceeded the predictive power of the first biceps burst to second triceps burst latency alone. The sequential order of biceps and triceps brachii activation was again shown to be a key mechanism in the prediction of Load 0 movement time to maximal displacement.

Under increased inertial loading, the predictive power of antagonist maximal isometric strength was revealed. Extension M.V.C. accounted for 67 percent of the variance associated with movement time to maximal displacement, under load conditions 1, and 65 percent of variance associated with movement time to maximal displacement, under load condition 2. The criterion measures selected second included: second triceps motor time, slope for the first biceps burst EMG, and first biceps burst to second triceps burst latency. However, second triceps motor time was selected second, whenever it remained available. Clearly the antagonist maximal force capability and activation pattern are important to the understanding of speed of human limb movement allowing multiple R's of .88 to .97 for the prediction of movement time under both unloaded and loaded conditions.

Full details of the above study can be found in the February 1983 Annual Report and in the Ph.D. dissertation "Gender Differences and the Effects of Isometric Fatigue and Relative Isometric Fatigue on the Maximum Speed of Human Forearm Flexion Under Resisted and Unresisted Conditions" by Zulma C. Garcia, Department of Exercise Science, University of Massachusetts-Amherst, 1983, 327pp. Sponsored by Walter P. Kroll.

## Effects of Isometric Muscular Fatigue and The Tonic Vibratory Response on The Speed of Forearm Flexion Movement In Women

The major purpose of this study was to assess the effects of isometric muscular fatigue and the tonic vibratory response (TVR) on the speed of forearm flexion movement in women. A secondary problem was to examine the relationship between the maximum isometric strength of the agonist and antagonist muscle groups, and the speed of movement.

Fifteen university women 18-26 years of age served as subjects. Subjects were tested on six days. The first two days were used to establish baseline measures of speed of forearm flexion movement, and maximum isometric strength of the forearm flexors and extensors. The four treatment days were balanced over subjects and across days, and consisted of two days of isometric muscular fatiguing exercise (one of the forearm flexors, and one of the extensors), and two days of tonic vibratory response treatment (one of the flexors, and one of the extensors).

The tonic vibratory response (TVR) is an involuntary contraction of vibrated muscle fiber, and is believed to be caused by excitation of the muscle spindle primary endings. Vibration of the forearm flexors causes a tonic contraction to occur, which is generally accompanied by an inhibition of the forearm extensors; the reverse facilitory-inhibitory relationship occurs when the forearm extensors are vibrated. The effects of the TVR has been observed to last up to 100 seconds following the removal of vibration. In view of these facts, a TVR elicited in the forearm flexors was expected to facilitate the forearm flexors and



the forearm extensors. This was expected to increase the speed of movement by increasing the torque produced by the forearm flexors relative to the forearm extensors. Facilitation of the forearm extensors was expected to lengthen movement time due to increased drag of the forearm extensors and decreased torque of the forearm flexors.

In order to test this hypothesis the forearm flexors and extensors, on separate days, were vibrated for 100 seconds at a frequency of 100-110 Hz to elicit a TVR. As this was a standard frequency of sufficient duration, it is assumed that a TVR was achieved in all subjects. A period of 100 seconds of vibration was immediately followed by five speed of forearm flexion movement trials, and this sequence was repeated four times.

Fatigue has been shown to have detrimental effects upon performance. The use of fatigue as an experimental treatment allows a comparison to be made between movement speed under normal and under fatigued conditions. Several studies have shown that agonist muscle fatigue results in longer movement times even though agonist strength does not correlate with movement time.

The results showed that the reliability coefficients ranged between .78 and .94 for the measures of movement time, acceleration time, percent acceleration time, and maximum isometric flexion and extension strength. Movement parameters did not demonstrate significant practice effects, and the baseline measures were extremely stable. Movement time was significantly correlated with percent acceleration time,  $r = -.90$ , ( $p < .01$ ). Movement time was not found to have a statistically signi-

ficant relationship with the maximum isometric strength of the forearm flexors or extensors.

Fatiguing isometric exercise of the forearm flexors produced a 35.9% decrement in maximum isometric forearm flexion strength from pre-to- post-treatment measures. The strength decrement was accompanied by a 29.8 ms lengthening of movement time, a 30.2 ms decrease in acceleration time, and a 25.8% drop in percent acceleration time. These changes in movement parameters were all significant at the .10 level. Fatiguing isometric exercise of the forearm extensors resulted in an 8.3% decrease in maximum isometric extension strength, which was a significant strength decrement at the .05 level. The fatiguing isometric exercise of the forearm extensors was not accompanied by any significant changes in the movement parameters.

The tonic vibratory response (TVR) treatment applied to the forearm flexors resulted in a significant lengthening of movement time ( $p < .05$ ), but did not significantly alter acceleration time, or percent acceleration time. The tonic vibratory response treatment applied to the forearm extensors did not produce significant differences in any of the movement parameters. Tonic vibratory response treatment applied to the forearm extensors produced no significant changes in any of the movement parameters. Surprisingly, maximum isometric extension strength was 12.3% lower following the four treatment sessions of extensor muscle vibration.

The tonic contraction of the muscle generally dissipates within a few seconds of removal of vibration, although the effects of the TVR on reflexive contractions last for up to 100 seconds of the post vibratory period. Perhaps the facilitatory effects of the TVR on the forearm extensors diminished too quickly to cause a significant decrease in

the speed of movement. It is also possible that voluntary muscular effort overrides any effect the TVR may have on the speed of the forearm flexion movement. The TVR is not a strong reflex, and subjects are generally able to prevent the contraction from occurring if provided with a visual readout of the tension. It seems likely that the strong voluntary contraction of the forearm flexors to produce a maximum speed of forearm flexion movement could overcome the deleterious effects of a TVR induced in the forearm extensors.

Full details of the above report can be found in the February 1982 Annual Report and in the Master's thesis "Effects of Isometric Muscular Fatigue and the Tonic Vibratory Response on the Speed of Forearm Flexion Movement in Women" by Marilyn A. Teves, Department of Exercise Science, University of Massachusetts-Amherst, 1981, 160pp. Sponsored by Walter P. Kroll.

### Quantitative Analysis of Practice Effects Upon the Triphasic EMG Pattern for a Maximum Speed Forearm Flexion Movement

Eight male and eight female subjects were tested for maximum speed forearm flexion movement under each of three different inertial load conditions on eight days. Daily alteration of two kinematic (movement time and percent acceleration time) and six raw EMG parameters were studied in detail. The six raw EMG parameters assessed were number of spikes (NOS), mean spike amplitude (MSA), mean number of peaks per spike (MNP), spike frequency (SF), mean spike duration (MSD), and mean spike slope (MSS). Four temporal components of the triphasic EMG pattern were also assessed: biceps brachii motor time (B1), end of the first biceps burst (B2), second biceps brachii burst (B3), and triceps brachii burst (B4).

Results showed that all quantitative EMG parameters (except mean spike slope) were measured reliably for biceps brachii B1 and B2 components. Practice effects produced faster movement times and similar EMG spike parameter changes in biceps brachii B1 and B2 components. Practice effects produced a decrease in mean spike duration (MSD) of 12 and 13 percent for biceps brachii B1 and B2 components, respectively. Spike frequency (SF), on the other hand, increased eight and eleven percent for B1 and B2 components. Inertial loading was associated with an increase in the number of spikes (NOS) in B1 and B2 components. Movement time correlated  $r = .57$  with number of spikes in the B2 component. Biceps brachii B2 component correlated negatively  $r = -.65$  with percent acceleration time (PAT).

This investigation was the first to measure reliably quantitative EMG parameter changes due to practice effects in fast ballistic limb movement. The mean decrease of 15 milli-seconds (10 percent) in forearm flexion movement time was associated with a shortening of the total biceps brachii burst, a 12 percent decrease in mean spike duration, and an eight to eleven percent increase in spike frequency. These results suggest that firing rate modulation is the major mechanism involved in motor unit firing pattern changes due to practice. Synchronization of motor unit firing or the recruitment of faster firing high threshold fast twitch motor units do not appear to be neuromotor coordination mechanisms involved in practice changes. Although higher threshold fast twitch motor units could produce a faster firing frequency, the reduction in mean spike duration negates such an explanation. Higher firing rates associated with fast twitch motor units would also produce longer spike durations which was not the case. Instead, the same motor unit pool is being recruited but made to fire at higher frequencies.

Full details of the above study can be found in the February 1983 Annual Report and in the Master's thesis "Maximum Speed of Forearm Flexion Practice Effects Upon Surface EMG Signal Characteristics" by Mark S. Flieger, Department of Exercise Science, University of Massachusetts-Amherst, 1983, 249pp. Sponsor: Walter P. Kroll.

## Patterned Electrical Stimulation Effects Upon Neuromuscular Coordination Control Mechanisms Underlying Speed of Forearm Flexion Movement

Thirty-six subjects were allocated equally to a control, a physical practice, and four different electrical stimulation experimental groups. Three pre-test stabilization days were followed by two treatment periods of two weeks duration with a post-test following each of the two week treatment periods. The four experimental groups were each administered with a different electrical stimulation protocol for six 30-minute sessions during each of the two week treatment periods. Each 30-minute session included an electrical stimulation protocol pattern being administered once every 10-seconds for a total of 180 patterns each session. Over six such sessions 1,080 electrical stimulation protocol patterns would be administered. In the second two-week treatment period another such 1,080 patterns would be administered.

The four electrical stimulation groups consisted of two high frequency and two low frequency groups. In both the high and the low frequency groups, a progression and a retrogression group also existed. Progression groups received a longer agonist to antagonist latency pattern while the retrogression groups received a shorter agonist to antagonist pattern. Since the agonist to antagonist latency parameter is one of the major neuromotor coordination mechanisms in fast limb movement it was hypothesized that manipulation of this pattern would produce modification in

the triphasic EMG pattern as well as in task performance. The low frequency (Hz 50) stimulation is within the normal neurophysiologic range of motor unit firing while the high frequency stimulation (1,000 Hz) is not. The question of employing normal rather than abnormal wave form characteristics in electrical stimulation would thus be tested.

Following the initial baseline stabilization practice days, movement time was significantly affected by the patterned electrical stimulation pattern protocols. The progression (longer agonist to antagonist latency) groups were found to be slower after two-week treatment periods while the retrogression groups got faster. Antagonist muscle activity, particularly the IEMG slope parameter, was significantly affected by the patterned electrical stimulation treatment periods. Except for a decrease in total EMG duration, agonist muscle activity parameters remained essentially unchanged. Prediction of movement speed was affected by practice effects. In the unpracticed condition, the agonist muscle activity was a more important predictor than the antagonist muscle. After extended practice, the antagonist muscle activity parameters were more important predictors.

Based upon neuromotor coordination mechanisms identified in previous studies, patterned electrical stimulation protocols were shown to be an effective technique with which to manipulate limb movement speed. Further, improvements in limb movement speed produced by patterned electrical stimulation were associated with

predicted changes in the triphasic EMG pattern. Such changes in the triphasic EMG pattern were effectively manipulated by the patterned electrical stimulation protocols.

Full details of the above study can be found in the February 1983 Annual Report and in the doctoral dissertation "Modelled Functional Electrical Stimulation Effects Upon Neuromuscular Coordination Control Mechanisms Underlying Speed of Human Movement" by Jean Paul Boucher, Department of Exercise Science, University of Massachusetts-Amherst, 1984, 354pp. Sponsor: Walter P. Kroll.



High Frequency Electrical Stimulation of Agonist and Antagonist  
Muscle Groups Involved in Fast Forearm Flexion Movement: Effects  
Upon Movement Time and the Triphasic EMG Pattern

Ten male and ten female subjects were allocated equally to two experimental groups. One group received electrical stimulation of the elbow flexion muscle group and the other group received electrical stimulation of the elbow extensor muscle group. Flexion and extension movement speed in unloaded and loaded conditions, EMG of biceps brachii and triceps brachii muscles during movement trials, maximum isometric strength, and endurance holding time with a load equal to 50 percent of maximum isometric strength were assessed over four baseline stabilization days. Following baseline days, 18 sessions of electrical stimulation were administered using a high frequency (2500 Hz) Russian type stimulator. Two post-test sessions duplicated baseline stabilization day measurements.

The main conclusion reached was that high frequency electrical stimulation did not produce the strength increases reported by Russian investigators. Males did show a 23 percent increase in extension strength in the arm which received stimulation on the flexors. Only a 12.3 percent increase in extension strength occurred in males when the extensors were stimulated. One probable explanation for the above results in which extension strength increased more with flexor stimulation than with extension stimulation is that the stimulated flexor contraction was so intense it required subjects to co-contract the extensors to project the joint. Such active

co-contraction produced greater strength increases than actual electrical stimulation protocols.

Movement time changes occurred mostly in the male subjects. Males receiving flexor stimulation had approximately a 5 percent faster flexion movement time in both unloaded and loaded conditions. Flexor stimulation in males also produced 12.4 and 12.9 faster extension movement times in unloaded and loaded conditions, respectively. Males received extensor stimulation showed 12.4 percent faster extension movement time. Females displayed differential changes in movement speed due to electrical stimulation and no significant increases in strength.

Full details of the above study can be found in the February 1983 Annual Report.

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